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Selection against migrants contributes to the rapid evolution of ecologically dependent reproductive isolation

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ABSTRACT

Ecologically dependent reproductive isolation (EDRI) is the decrease in gene flow that occurs when adaptive divergence reduces the fitness of individuals that move between ecological environments. A particularly simple and perhaps almost universal mechanism contributing to EDRI is natural selection against migrants themselves (i.e. before any selection against their offspring). Here I use single-locus and quantitative-genetic models to examine how quickly this mechanism can contribute to EDRI after the founding of a new population. I find that as long as divergent selection is relatively strong, selection against migrants can make a substantial contribution to EDRI in less than 30 generations. This mechanism is only one of several that might influence EDRI. The addition of other mechanisms, such as habitat choice or selection against hybrids, would presumably accelerate the accumulation of EDRI. Although EDRI is neither complete (some gene flow may still occur) nor unconditional (it depends on the ecological context), it may reduce gene flow to the point that absolute and unconditional reproductive isolation can evolve through other mechanisms. Whenever organisms colonize new ecological environments, selection against migrants may contribute to rapid reductions in gene flow. Future studies of ecological speciation may profit from particular attention to this mechanism of reproductive isolation.

Keywords: adaptive divergence, contemporary evolution, ecological speciation, gene flow, rapid evolution, reinforcement, reproductive isolation.

INTRODUCTION

The potential mechanisms by which one species can split into two are many – for example, mutation and genetic drift, natural and sexual selection, hybridization, polyploidy, and chromosome rearrangements (Dobzhansky, 1951; Mayr, 1963; Coyne, 1992; Schluter, 2000; Barton, 2001; Gavrilets, 2003). Of these mechanisms, natural selection has received an increasing amount of attention and is now considered one of the primary causes of speciation in animals (Rice and Hostert, 1993; Orr and Smith, 1998; Schluter, 2000). One

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way in which natural selection contributes to speciation is by causing the adaptive divergence of populations inhabiting different ecological environments, which then reduces gene flow as a byproduct (Darwin, 1859; Dobzhansky, 1951; Mayr, 1963; Schluter, 2000). Adaptive divergence can generate both pre-zygotic and post-zygotic isolation, which in either case might or might not depend on the environmental context (i.e. 'conditional' *vs* 'unconditional'; Rice and Hostert, 1993). Here I focus on conditional reproductive isolation that arises because fitness is higher for individuals breeding in environments to which they are adapted than in environments to which they are not adapted. This 'ecologically dependent reproductive isolation' (EDRI) has the potential to play an important role in speciation (Schluter, 2000; Rundle and Whitlock, 2001).

I am particularly interested in how quickly reproductive isolation arises when organisms colonize and adapt to new environments. On the empirical side, laboratory studies that impose strong divergent selection often generate substantial EDRI in a very short time (Rice and Hostert, 1993), but these studies may impose strengths of selection that are rare in nature. For natural populations, many examples of EDRI have been advanced (Schluter, 2000), but only a few are known to have arisen over a reasonably short time. First, three-spine stickleback (*Gasterosteus aculeatus*) that began adapting to different resources (benthic *vs* limnetic in lakes) about 5000 generations ago now show almost no gene flow in sympatry (Schluter, 2000; Taylor and McPhail, 2000; Hendry *et al.*, 2001). Second, phytophagous insects that colonized new host plants 100–200 generations ago now exchange few genes with their sympatric ancestors (Feder *et al.*, 1988, 1994; Via, 1999; Via *et al.*, 2000). Third, introduced sockeye salmon (*Oncorhynchus nerka*) that began adapting to different breeding environments (lake beach *vs* river) 12–14 generations ago now show limited gene flow despite high rates of adult mixing (Hendry *et al.*, 2000; Hendry, 2001). In each of these cases, intrinsic genetic incompatibilities are absent and reproductive isolation appears to be ecologically dependent.

On the theoretical side, many studies have shown that divergent selection can cause substantial reproductive isolation, sometimes in fewer than 100 generations (e.g. Orr and Orr, 1996; Gavrilets *et al.*, 1998; Dieckmann and Doebeli, 1999; Kondrashov and Kondrashov, 1999; Gavrilets, 2000, 2003; Church and Taylor, 2002; Porter and Johnson, 2002; Doebeli and Dieckmann, 2003). These models incorporate a variety of isolating mechanisms, the most common being unconditional genetic incompatibilities, competition in relation to phenotypic similarity, ecological selection against hybrids, and assortative mating. Although these models have greatly improved our understanding of speciation, they are usually difficult to apply to natural systems. In the present paper, I develop models that evaluate the effects of a particularly simple, powerful and common contributor to EDRI. Specifically, individuals adapted to one environment that move to a different environment (i.e. 'migrants') will be maladapted and should therefore have reduced survival or reproductive performance.

This 'selection against migrants' is rarely invoked in the context of reproductive isolation, and has not been evaluated in theoretical models. And yet this mechanism should be a powerful force reducing gene flow among diverging populations (e.g. Nagy and Rice, 1997; Cooper, 2000; Via *et al.*, 2000; Nosil, 2004; Nosil *et al.*, in press). The potential importance of selection against migrants has been particularly well documented in work on phytophagous insects, one of the systems in which EDRI appears to have arisen quickly. Via *et al.* (2000) reciprocally transplanted pea aphids (*Acyrthosiphon pisum*) between two host plants (alfalfa and clover) and monitored their subsequent survival and fecundity. The fitness of alfalfa

aphids transplanted to clover was 6% that of clover aphids on clover ('clover residents') and 4.6% that of alfalfa aphids on alfalfa ('alfalfa residents'). Similarly, the fitness of clover aphids transplanted to alfalfa was 0.8% that of alfalfa residents and 1% that of clover residents. Via *et al.* (2000) also demonstrated that *natural* migrants between alfalfa and clover have lower fitness than residents on each host plant. Similarly, Nosil (2004) has found that selection against migrants may be more important than mate choice in establishing pre-mating isolation between walking-stick insects (*Timema cristinae*) adapted to different host plants. More generally, the large number of reciprocal transplant studies that demonstrate higher performance in 'home' than 'foreign' environments can all be interpreted as providing indirect evidence for the importance of selection against migrants (Via *et al.*, 2000).

In the present paper, I use single-locus and quantitative-genetic models to determine how quickly selection against migrants can contribute to EDRI. In these analyses, I track the temporal change in gene flow between populations that colonize and adapt to different environments. I show that this change is a function of several different effects, only some of which should be considered a part of EDRI. I therefore specifically track EDRI as the average per-generation genetic contribution of a migrant, relative to a resident, within an adapting population. In all cases, I assume outbred populations so that heterosis does not increase the fitness of migrants (Ingvarsson and Whitlock, 2000; Ebert *et al.*, 2002). I find that selection against migrants can make a substantial contribution to EDRI in less than 30 generations. This result is robust even in the presence of substantial on-going physical mixing between environments (e.g. movement of adults), as long as divergent selection is reasonably strong.

ANALYTICAL RESULTS

I model gene flow from an ancestral continent population into a newly founded island population. In this scenario, gene flow into the island population will be a function of the number and fitness of migrants relative to residents. To isolate the effect of selection against migrants, I consider only fitness differences that are manifested before zygotes are produced; afterwards, any offspring are considered part of the island population. Gene flow from the continent to the island is tracked as the per-generation (discrete generations) genetic contribution of migrants relative to residents (φ):

$$\varphi = \frac{MN\overline{W}_M}{MN\overline{W}_M + n\overline{W}_R} \tag{1}$$

The numerator of this equation represents the number of genes that migrants contribute to the next island generation, whereas the denominator represents the total number of genes both migrants and residents contribute. The number of genes that migrants contribute is simply the number of migrants (i.e. probability that a continent individual will migrate to the island, M, multiplied by number of continent individuals, N) multiplied by the average absolute fitness of a migrant ($\overline{W}_M$). The total number of genes that both migrants and residents contribute is the number of migrant genes ($MN\overline{W}_M$, as above) plus the number of resident genes, the latter being the number of resident individuals (n) multiplied by their average absolute fitness ($\overline{W}_R$). (For diploid organisms, each term is multiplied by two, which then cancels.)

Most evolutionary models ignore population dynamics (Holt and Gomulkiewicz, 1997) and consider gene flow as a temporally constant proportion of the island population – that is, $m = MN/(MN + n)$. In this case, equation (1) becomes

$$\varphi = \frac{m\overline{W}_M}{m\overline{W}_M + (1-m)\overline{W}_R} \tag{2}$$

To consider the *rate* at which gene flow changes with time, I examine a case where migrants from the continent found a new island population, which then adapts to the island environment. In this case, island population size (n), and hence m, should change through time. I therefore retain equation (1) but here rearrange it to better illustrate the factors that influence gene flow – that is, the number of migrants relative to residents (MN/n) and the fitness of migrants relative to residents ($\overline{W}_M/\overline{W}_R$):

$$\varphi = \frac{1}{1 + \frac{1}{\left(\frac{MN}{n}\,\frac{\overline{W}_M}{\overline{W}_R}\right)}} \tag{3}$$

This equation illustrates four ways in which gene flow can change following the colonization of a new environment.

1. Island population size

If the continent population size (N) and the probability that a continent individual moves to the island (M) both remain constant, the *number* of individuals moving from the continent to the island (MN) will also remain constant. The island population, however, should grow from a founding number of individuals ($n = MN$) to some larger equilibrium size. The *proportion* of individuals on the island that are migrants (m) should therefore decrease through time until the equilibrium is reached. This temporal decrease in proportional gene flow will occur even if (i) migrants and residents have equal fitness (i.e. when $\overline{W}_M/\overline{W}_R = 1$, $\varphi = MN/(MN + n)$), (ii) the continent and island populations occupy identical environments (i.e. no difference in selection), or (iii) no genetic polymorphism is present. This population size effect is therefore not the direct result of adaptation and should not be considered a part of EDRI. It is possible, however, that adaptation may increase equilibrium population size and therefore have an indirect influence on gene flow through this population size effect.

2. Adaptation without density dependence

When the continent and island populations occupy different ecological environments, selection should cause their adaptive divergence. As the island population adapts to its new environment, the fitness of island residents should increase relative to the fitness of migrants, which remain adapted to the continent. One context for this change in relative fitness occurs when density dependence and migrant–resident interactions are weak, which might be the case during the initial stages of population divergence. Under these conditions, the absolute fitness of migrants might remain constant, whereas the absolute

fitness of residents should increase. The resulting decrease in the *relative* fitness of migrants should reduce gene flow even in the absence of changes in island population size – that is, if M, N, $\overline{W}_M$ and n are all constant, $\varphi = 1/(1 + const(\overline{W}_R))$. This density-independent reduction in gene flow contributes to EDRI because it results directly from adaptation and because it depends on the environmental context.

3. Adaptation with density dependence

Increasing island population size should initiate density dependence and have a negative effect on absolute fitness, which may influence gene flow if residents and migrants respond differently to density. For example, all individuals might be successful at obtaining high-quality feeding or breeding sites when population densities are low. At high densities, however, residents may obtain better sites than migrants because residents are better adapted for competition in their home environment. This density-dependent reduction in gene flow contributes to EDRI because it results directly from adaptation and depends on the environment.

Note that absolute fitness responds differently to density-independent versus density-dependent adaptation. When density dependence is weak (early in divergence), the absolute fitness of migrants might remain constant through time, whereas the absolute fitness of residents should increase. When density dependence becomes important (later in divergence), the absolute fitness of both residents and migrants should decrease. If, adaptation causes density dependence to act less strongly on residents than migrants, the decline in fitness should be less for residents.

4. Interactions between population size and adaptation

Higher fitness of residents than migrants, which can occur owing to adaptation with or without density dependence, will interact with island population size to influence overall gene flow (Fig. 1). In some situations, migrants may represent a substantial proportion of

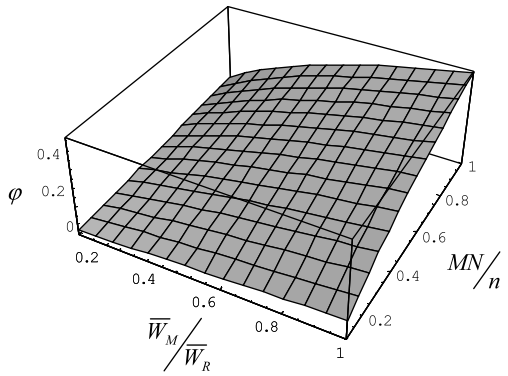

Fig. 1. The rate of gene flow (φ) from the continent into the island for various ratios of the number of migrants to residents (MN/n) and the fitness of migrants to residents ($\overline{W}_M/\overline{W}_R$). The graph is based on equation (3), and shows that gene flow is lower when migrants are rare relative to residents and when migrant fitness is low relative to resident fitness. It also shows that the effect of $\overline{W}_M/\overline{W}_R$ on reducing gene flow decreases as MN/n decreases.

the island population, such as when the number of migrants is large (large continent population or high probability of movement to the island) or the island population is small (early in divergence or when the island carrying capacity is low). In other situations, migrants may represent a small proportion of the island population, such as under the opposite conditions: small continent population, low probability of migration, late in divergence, or high island carrying capacity. In the first situation, a given decrease in the relative fitness of migrants will have a larger effect on reducing overall gene flow. In the second situation, the same relative fitness difference will have a smaller effect on reducing overall gene flow.

Conclusions and predictions

Gene flow (φ) is influenced by changes in island population size, the fitness of residents and migrants, and the interaction between population size and relative fitness. Of these effects, reductions in gene flow that result directly from adaptation are a part of EDRI, which can therefore be indexed as the ratio of migrant to resident fitness ($\overline{W}_M/\overline{W}_R$). The magnitude of this ratio will depend on adaptation (with and without density dependence) but not on the simple numerical effect of changes in population size. I therefore use (1) $\overline{W}_M/\overline{W}_R$ to infer how selection against migrants contributes to EDRI, (2) the effect of $\overline{W}_M/\overline{W}_R$ on φ to infer how selection against migrants influences overall gene flow, and (3) φ to infer how overall gene flow changes through time.

Several general conclusions emerge. First, adaptive divergence should cause a decrease in the fitness of migrants relative to residents, which represents the contribution of selection against migrants to EDRI. Second, EDRI should increase most rapidly early in population divergence. One reason for this expectation is that a given absolute change in $\overline{W}_R$ will cause a greater proportional change in $\overline{W}_M/\overline{W}_R$ when this ratio is close to unity (early in divergence) than when it is small (late in divergence). Another reason is that density-independent adaptation should be most rapid during the initial stages of divergence – because this is when selection is strongest. This expectation may be counteracted, however, by density-dependent adaptation, which should change most rapidly after the island population becomes larger. Third, the effect of a given amount of EDRI on overall gene flow should be greatest when the island population is small relative to the number of migrants – because a given decrease in $\overline{W}_M/\overline{W}_R$ will have a greater absolute impact on φ when MN/n is high than when it is low (Fig. 1).

SIMULATIONS

The above analytical results provide qualitative predictions for how population size and selection against migrants should influence gene flow. They cannot, however, provide a quantitative answer to the question of most interest here: How quickly does selection against migrants contribute to EDRI and reduce the overall amount of gene flow? To answer this question, I employ single-locus and quantitative-genetic models to simulate the divergence of a newly founded island population from its continental ancestor (model details are provided in the Appendix).

For each simulation, MN individuals from the continent are used to found a new island population in the first generation. This new population then adapts to the island environment and increases in abundance to eventually asymptote at the island carrying capacity. For simplicity, I assume temporal constancy in the continent population,

including population size (N), allele frequency (single-locus model) or mean phenotype (quantitative-genetic model), and probability of migration to the island (M). I further assume that migrants are always typical of continent residents (in allele frequency or phenotypic trait distribution) and that migration from the island back to the continent is absent. Relaxing these assumptions would increase model complexity without appreciably influencing the general conclusions. For example, ongoing selection on the continent that differed from the island would probably speed the rate at which EDRI evolves.

The simulations assume a discrete-generation life cycle where migration occurs first, then selection takes place, and then reproduction occurs. After reproduction (i.e. the next generation), any descendents of migrants are fully integrated into the resident gene pool (as in the analytical model). This approach isolates the effect of selection against migrants from any selection that might act on their offspring. My primary goal in the simulations was to determine the time scale over which selection against migrants contributes to EDRI and reduces gene flow. I therefore present results through generation 30 only because most of the changes occurred during that interval. Although it is possible, at least in principle, to obtain analytical solutions for equilibrium conditions, I have not done so because the present analysis is concerned with model dynamics rather than equilibria.

Single-locus model

I used the model outlined in the Appendix to explore the effects of various combinations of continent population size (N), probability of a continent individual migrating to the island (M), frequency of allele A on the continent and in migrants (P), density-independent fitness of genotype ij (x_{ij}), and strength of density dependence acting on genotype ij (y_{ij}). Selection acts on two alleles (A and a), one of which (A) is favoured on the island (i.e. $x_{AA} \geq x_{aa}$ and $y_{AA} \leq y_{aa}$) and is present in some constant frequency on the continent. The results hold, however, regardless of the specific allele that is favoured on the island.

The model generated plausible changes in island population size (n), frequency of the favoured allele on the island (p), resident fitness ($\overline{W}_R$), migrant fitness ($\overline{W}_M$), EDRI ($\overline{W}_M/\overline{W}_R$), and gene flow ($\varphi$). Figure 2 shows the temporal progression of these quantitites for a typical set of parameter values. In general, island population size first increased slowly and then rapidly, eventually levelling off at a carrying capacity determined by the density-independent and density-dependent fitness of each genotype (results not shown). The frequency of the favoured allele increased towards unity at a rate that depended primarily on the fitness difference between homozygous genotypes. Resident fitness started at the average fitness of migrants at low density, and then (1) increased as island residents became better adapted (i.e. an increase in the frequency of the favoured allele), (2) decreased owing to density dependence, and (3) equilibrated at almost unity (exactly unity if migration ceased). The transitory nature of this increase in resident fitness confirms intuition that density-independent adaptation is most important during the intitial stages of population divergence. Migrant fitness declined slowly at first and then more rapidly as density dependence became important. EDRI followed a pattern similar to migrant fitness, but initially decreased at a faster rate owing to the effect of density-independent adaptation. Gene flow (φ) decreased rapidly but at a decelerating rate, a pattern largely reflecting the increase in island population size and the resulting decrease in migration rate (m).

Selection against migrants made a substantial contribution to EDRI in less than 30 generations (Fig. 3). The maximum amount of EDRI and the rate at which it arose were

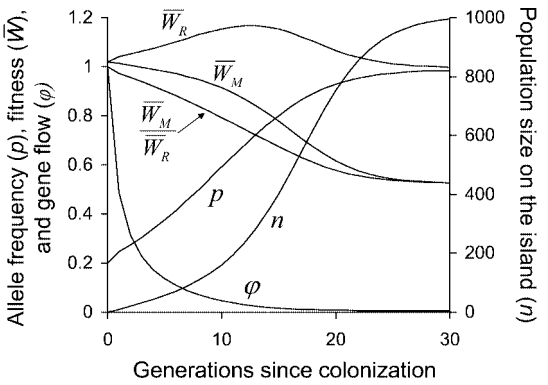

Fig. 2. For the single-locus model, a typical temporal progression of island population size (n), frequency of the favoured allele (p), migrant fitness ($\overline{W}_M$), resident fitness ($\overline{W}_R$), EDRI ($\overline{W}_M/\overline{W}_R$), and gene flow ($\varphi$). For this representative simulation, the number of migrants was $MN = 10$, density-independent fitnesses were $x_{AA} = 1.5$ and $x_{aa} = 0.9$, and density-dependent effects were $y_{AA} = 0.0004$ and $y_{aa} = 0.0008$.

functions of (1) the allele frequency on the continent (EDRI was stronger when the allele favoured on the island was rarer on the continent), (2) the fitness of different genotypes in the absence of density dependence (EDRI was stronger when x_{AA}/x_{aa} was larger), and (3) the fitness of different genotypes in the presence of density dependence (EDRI was stronger when y_{AA}/y_{aa} was smaller). EDRI was relatively insensitive to the number of migrants (MN), at least until MN exceeded approximately 10% of the equilibrium island population size (results not shown), after which EDRI decreased but remained substantial. For example, when $MN = 10$, the equilibrium $n = 1008$ and the equilibrium $\overline{W}_M/\overline{W}_R = 0.527$; whereas when $MN = 500$, the equilibrium $n = 1019$ and the equilibrium $\overline{W}_M/\overline{W}_R = 0.772$ (for both cases: $P = 0.2$, $x_{AA} = 1.5$, $x_{aa} = 0.9$, $y_{AA} = 0.0004$, $y_{aa} = 0.0008$). In all cases, selection against migrants did not cause EDRI if the continental population was fixed for the allele that was favoured on the island (Fig. 3).

In the above simulations, allele A was assumed to be favoured on the island when densities were both low ($x_{AA} \geq x_{aa}$) and high ($y_{AA} \leq y_{aa}$). It is worth noting, however, that transitory increases and decreases in EDRI can occur when one allele is favoured at low densities but the other is favoured at high densities. Whether or not such conditions are commonly encountered in nature requires empirical investigation.

Quantitative-genetic model

I used the model outlined in the Appendix to explore the effect of various combinations of continent population size (N), probability of a continent individual migrating to the island (M), deviation of the mean phenotype on the continent from the optimal phenotype on the island ($\bar{Z} - \theta$), strength of density-independent stabilizing selection around the optimum (ω), additive genetic variance (G), fitness of a perfectly adapted individual in the absence of density dependence (α), strength of density dependence (c), and the effect of density on the fitness of an individual with a given phenotypic deviation from the optimum (k). This last parameter is important because it allows density to have different effects on individuals

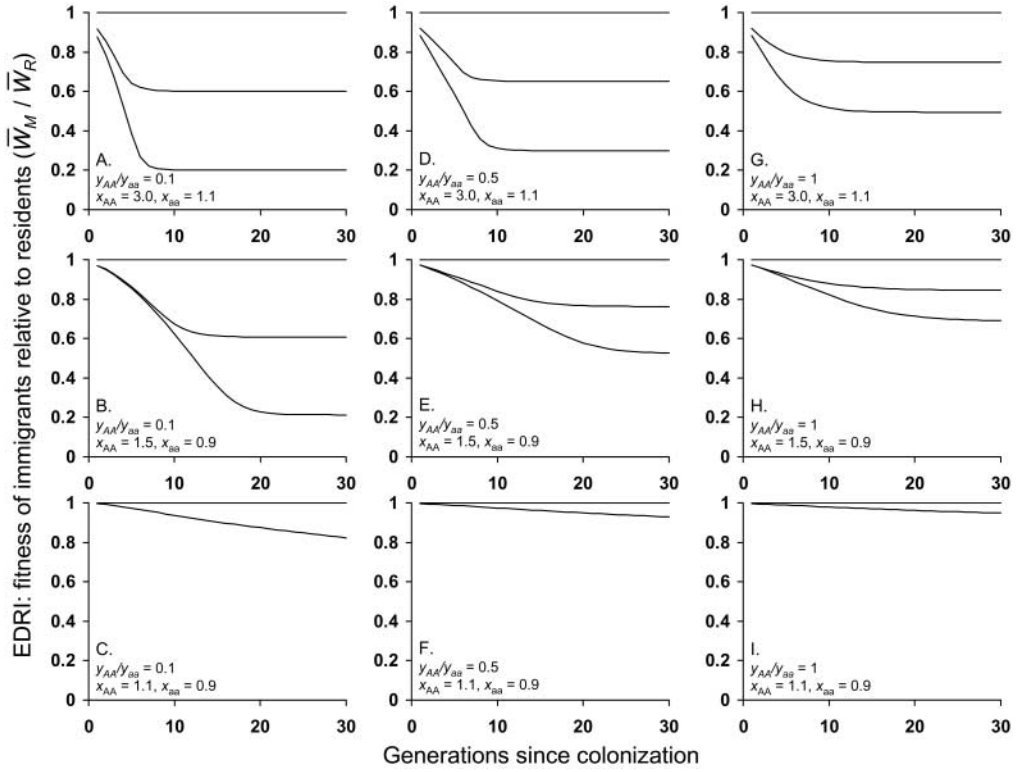

Fig. 3. For the single-locus model, the evolution of EDRI ($\overline{W}_M/\overline{W}_R$) owing to selection against migrants. Different lines in each panel show the effect of variation in the continent allele frequency: $P = 1.0$, 0.6 and 0.2 (from top to bottom) for the panels with three lines and $P = 1.0$ and 0.2 for panels with two lines ($P = 0.6$ not shown for clarity). Different columns of panels show the effect of variation in the strength of density dependence acting on homozygous genotypes ($y_{AA}/y_{aa} = 0.1$, 0.5 and 1.0). Different rows of panels show the effect of various combinations of density-independent genotype fitnesses ($x_{AA} = 3.0$ and $x_{aa} = 1.1$; $x_{AA} = 1.5$ and $x_{aa} = 0.9$; and $x_{AA} = 1.1$ and $x_{aa} = 0.9$). For all cases, $MN = 10$.

with different phenotypes, which is the case for migrants versus residents once adapation commences. I generally assume that $\overline{Z} - \theta < 0$, but the conclusions hold if $\overline{Z} - \theta > 0$.

The model typically yielded patterns (Fig. 4) similar to those observed for the single-locus model. In general, island population size (n) first increased slowly and then rapidly, eventually levelling off at a carrying capacity that depended on $\overline{Z} - \theta$, ω, α, c and k (results not shown). The mean phenotype on the island first increased rapidly and then asymptoted towards an equilibrium that depended on these same parameters (results not shown). If initial density-independent maladaptation was high, resident fitness increased until density dependence became important, after which it decreased. As in the single-locus model, this early but transitory increase in resident fitness confirms intuition that density-independent adaptation is important during the intitial stages of population divergence. If density-independent maladapation was low, resident fitness remained relatively constant before decreasing when density dependence became important. Resident fitness eventually

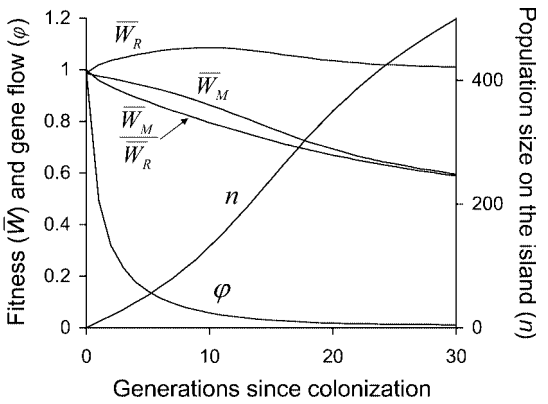

Fig. 4. For the quantitative-genetic model, a typical temporal progression of island population size (n), migrant fitness ($\overline{W}_M$), resident fitness ($\overline{W}_R$), EDRI ($\overline{W}_R$), and gene flow (φ). For this representative simulation, the number of migrants was $MN = 10$, the mean phenotype on the continent was $\bar{Z} = 1$, the optimal phenotype on the island was $\theta = 4$, the strength of density-independent stabilizing selection was $\omega = 4$, the additive genetic variance was $G = 0.4$, the phenotypic variance was $V = 1$, the fitness of a perfectly adapted individual in the absence of density dependence was $\alpha = 2.5$, the strength of density dependence was $c = 1000$, and the effect of population density on the fitness of a maladapted individual was $k = 1000$.

equilibrated near unity (exactly unity if migration ceased). Migrant fitness started at the same level as resident fitness and then decreased owing to density dependence. EDRI followed a pattern similar to migrant fitness but decreased more rapidly in simulations where resident fitness initially increased owing to density-independent adaptation. Gene flow (φ) decreased rapidly but at a decelerating rate, a pattern largely reflecting the increase in island population size and the resulting decrease in migration rate (m).

The contribution of selection against migrants to EDRI depended on several parameters, which I illustrate here for two plausible scenarios. In the first scenario, a given phenotypic deviation from the optimum ($\bar{Z} - \theta$) has relatively weak density-dependent effects (e.g. $k = 1000$) but relatively strong density-independent effects ($\omega = 2$–6). In this scenario, substantial EDRI usually arose in less than 30 generations (Fig. 5), with the maximum amount increasing as $|\bar{Z} - \theta|$ increased. The rate at which EDRI arose increased as the strength of density-independent stabilizing selection increased (i.e. ω decreased). Increasing additive genetic variance (G) had little effect on the maximum amount of EDRI but did increase the rate at which it arose (Fig. 5). In the second scenario, a given phenotypic deviation from the optimum had relatively strong density-dependent effects ($k = 10$–100) but relatively weak density-independent effects (e.g. $\omega = 20$). In this scenario also, substantial EDRI usually arose in less than 30 generations (Fig. 6), with the maximum amount increasing as $|\bar{Z} - \theta|$ increased. Additive genetic variance again influenced how quickly EDRI arose. As in the single-locus model, the number of migrants had minimal effects on the magnitude of EDRI and the rate at which it arose (results not shown). In all cases, selection against migrants did not contribute to EDRI if the mean phenotype on the continent was the same as the optimal phenotype on the island.

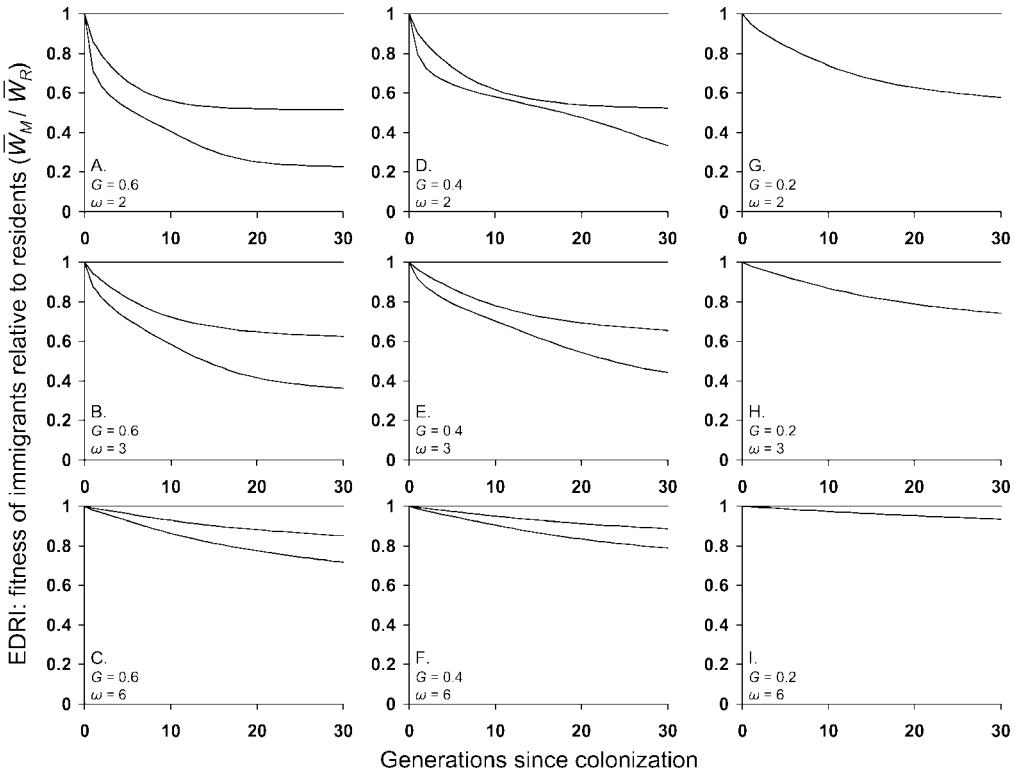

Fig. 5. For the quantitative-genetic model, the evolution of EDRI ($\overline{W}_M/\overline{W}_R$) owing to selection against migrants in the case of *strong* density-independent and *weak* density-dependent effects of maladaptation on fitness. Different lines in each panel show the effect of a given deviation of the mean continent phenotype from the island optimum: $\bar{Z} - \theta = 0$, –2 and –3 (from top to bottom) for panels with three lines and $\bar{Z} - \theta = 0$ and –2 for the panels with two lines (the island population was sometimes not sustainable at $\bar{Z} - \theta = -3$). Different columns of panels show the effect of additive genetic variance ($G = 0.6$, 0.4 and 0.2). Different rows of panels show the effect of density-independent stabilizing selection ($\omega = 2$, 3 and 6). For all cases, $MN = 10$, $V = 1$, $\alpha = 2.5$, $c = 1000$ and $k = 1000$.

DISCUSSION

The main conclusion is that selection against migrants can cause substantial EDRI in less than 30 generations. Reassuringly, the most important ingredient in this mechanism is also the most obvious and defensible: strong divergent selection between different ecological environments. The *maximum* amount of EDRI owing to selection against migrants was largely determined by the magnitude of the ecological difference between environments. The *rate* at which EDRI arose was also a function of the ecological difference, as well as the strength of density dependence and the amount of genetic variation.

The applicability of my results to natural systems was evaluated by comparing rates of phenotypic change in the quantitative-genetic simulations to rates observed in nature. Kinnison and Hendry (2001) compiled 2151 rates of microevolution, many from studies of populations adapting to new environments, and found that the upper 90th percentile over 30 generations was about two standard deviations (their figure 4a). The amount of

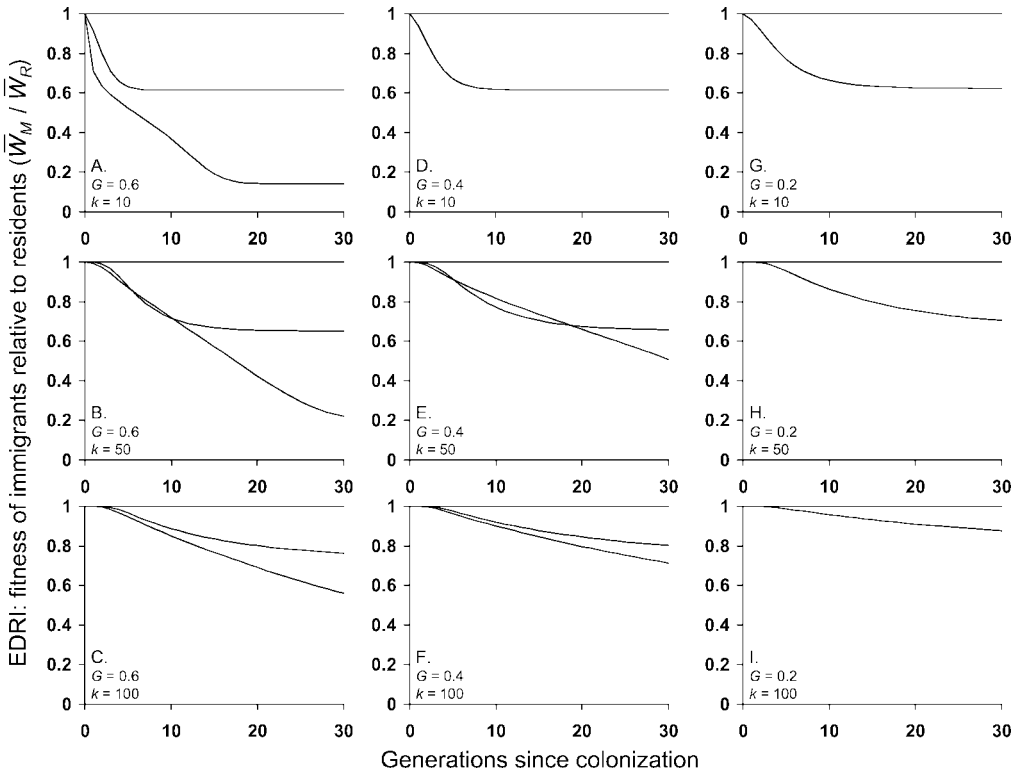

Fig. 6. For the quantitative-genetic model, the evolution of EDRI ($\bar{W}_M/\bar{W}_R$) owing to selection against migrants in the case of *weak* density-independent and *strong* density-dependent effects of maladaptation on fitness. Different lines in each panel show the effect of a given deviation of the mean continent phenotype from the island optimum: $\bar{Z} - \theta = 0$, −1 and −2 (from top to bottom) for the panels with three lines and $\bar{Z} - \theta = 0$ and −1 for the panels with two lines (the island population was sometimes not sustainable at $\bar{Z} - \theta = -2$). Different columns of panels show the effect of additive genetic variance ($G = 0.6$, 0.4 and 0.2). Different rows of panels show the effect of density-dependent stabilizing selection ($k = 10$, 50 and 100). For all cases, $MN = 10$, $V = 1$, $\alpha = 2.5$, $c = 1000$ and $\omega = 20$.

phenotypic change for each of the parameter combinations shown in Figs. 5 and 6 was less than two standard deviations in all but the most extreme cases: the lower line in the upper left panel of Fig. 5 (2.59 SD) and the lower line in the middle left panel of Fig. 5 (2.05 SD). These simulations thus generated rates of evolutionary change typical of natural populations.

The expectation that substantial EDRI can arise very quickly has considerable empirical support from laboratory experiments (Rice and Hostert, 1993) and natural populations (e.g. Feder *et al.*, 1988; Via, 1999; Hendry *et al.*, 2000; Via *et al.*, 2000). My single-locus (Fig. 3) and quantitative-genetic (Figs. 5 and 6) models show that selection against migrants could theoretically explain these findings. This conclusion is consistent with empirical work suggesting that this mechanism may be an important contributor to reproductive isolation in nature (e.g. Nagy and Rice, 1997; Cooper, 2000; Via *et al.*, 2000; Nosil, 2004; Nosil *et al.*, in press). Future studies of incipient speciation would benefit from examining the potential role of selection against migrants. Indeed, theoretical and empirical studies would ideally

examine the independent and synergistic effects of multiple ecological isolating mechanisms, including assortative mating, selection against migrants and selection against hybrids.

Selection against migrants is unlikely to generate reproductive isolation that is complete (i.e. zero gene flow) or unconditional (i.e. independent of the ecological context). And yet this mechanism is an effective way in which gene flow can be reduced repeatedly, dramatically and quickly. When organisms colonize different environments, some adaptation should be possible even if initial gene flow is high (Hendry *et al.*, 2001). This initial adaptation should lead to lowered fitness in migrants relative to residents, which will then further reduce gene flow (present study). This additional reduction in gene flow will allow further adaptive divergence, which will further reduce gene flow, and so on in a positive feedback loop. If the environments are sufficiently different, this iterative process may reduce gene flow to very low levels, which might then allow the evolution of complete and unconditional isolation through mechanisms that would otherwise be hindered by gene flow.

Another way in which selection against migrants may play an important role in speciation is by selecting for behaviours that reduce the probability of mating between individuals from different environments. Several such behaviours are possible. First, mate choice may evolve to reduce mating between individuals from different environments (e.g. 'reinforcement': Higgie *et al.*, 2000; Kirkpatrick, 2001; Nosil *et al.*, 2003). Second, habitat preferences may evolve to favour environments to which individuals are adapted (e.g. host plant preference: Rice, 1984; Bush, 1994; Via, 1999). Third, philopatry may evolve to ensure that individuals remain in (or return to) a specific breeding location (Hendry *et al.*, 2004). However, unlike the positive feedback loop described in the preceding paragraph, an initial reduction in gene flow owing to any of these behaviours would reduce the intensity of selection favouring further refinement of those behaviours.

I now return to an under-appreciated way in which gene flow can decrease very rapidly between diverging populations: the simple numerical effect of an increase in population size at the new location. As long as the island population size increases more rapidly than the number of migrants, which seems likely, the per-generation genetic contribution of migrants should decrease until an equilibrium is reached. This population size effect can lead to very low gene flow in a short period of time (Figs. 2 and 4), as long as the number of migrants is small relative to the equilibrium population size. This effect should not be considered a part of EDRI because it does not depend on adaptation, although it might be enhanced by adaptation that increases equilibrium population size.

The relative contributions of population size and EDRI to overall reductions in gene flow will depend on population size relative to the number of migrants (assume $NM = 10$ for the following). When the island population size is small (e.g. $n = 100$), then the reduction in gene flow ($1 - \varphi$) owing to the population size effect alone will be 90.9%. If the relative fitness of migrants is one-tenth that of residents ($\overline{W}_M/\overline{W}_R = 0.1$), the additional reduction in gene flow owing to EDRI will be 8.1% (total $1 - \varphi = 99\%$). This difference (90.9% *vs* 99%) could have large effects on the potential for evolutionary diversification. When the island population size is large (e.g. $n = 1000$), the reduction in gene flow owing to the population size effect alone will be 99%. If the relative fitness of migrants remains one-tenth that of residents, the additional reduction in gene flow owing to EDRI will be 0.9% (total $1 - \varphi = 99.9\%$). In this case, selection against migrants may have little additional impact on the potential for evolutionary diversification. The relative prevalence of these two scenarios in nature is not known, but the former appears likely for at least two cases in which EDRI appears to have

evolved rapidly. In pea aphids, physical dispersal between hosts is about 9–11% (Via, 1999). In introduced sockeye salmon, about 39% of the 'island' population (lake beach) was comprised of migrants from the 'continent' population (river), simply because the continent population was two orders of magnitude larger (Hendry *et al.*, 2000; Hendry, 2001). In these cases at least, EDRI has the potential to play a large role in reducing overall gene flow.

The relevance of the present results to natural systems will depend on the geographic context (allopatry, parapatry or sympatry). The model itself is perhaps best cast as 'allopatry with gene flow' because the populations were geographically distinct but still exchanged some migrants. This is likely to be a very common situation for speciation in nature. The models' qualitative results are probably also relevant to parapatric contexts, particularly when geographic structure is absent *within* each environment. When this is not true, such as in isolation-by-distance, clinal models should be more appropriate (e.g. Endler, 1977; Lande, 1982). The present results are least applicable to a purely sympatric scenario because the present geographic separation imposed an initial constraint on mixing between environments. Moreover, the present model lacked the frequency-dependent interactions that generate disruptive selection in models of adaptive sympatric speciation (e.g. Doebeli and Dieckmann, 2003). Nevertheless, selection against migrants (considered broadly to include individuals attempting to use environments to which they are not adapted) should be a potent force in any of these geographic contexts.

Conclusion

Selection against migrants may be one of the most prevalent forms of reproductive isolation in nature (Nagy and Rice, 1997; Cooper, 2000; Via *et al.*, 2000; Nosil, 2004; Nosil *et al.*, in press), simply because divergent selection between ecological environments is widespread (Schluter, 2000). Thus, examples from natural populations of substantial EDRI evolving after less than 30 generations (e.g. Hendry *et al.*, 2000; Hendry, 2001; Sheldon and Jones, 2001) should not be viewed as exceptional. Instead, they should be viewed as the expected outcome of a process that occurs when organisms colonize new environments. Once the baseline expectation has changed for how quickly this form of reproductive isolation can evolve, more investigators are likely to test for its presence. I expect these endeavours will reveal many other cases in which EDRI has evolved quickly. Moreover, the true beauty of EDRI is that it arises so quickly we might even be able to watch it evolve.

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APPENDIX

Single-locus model

Population size on the island changes from one generation (t) to the next ($t + 1$) according to

$$n_{t+1} = \overline{W}_R n + \overline{W}_M MN \tag{A1}$$

where the island population size (n), the continent population size (N), the probability that a continent individual migrates to the island (M), and the mean fitness of residents ($\overline{W}_R$) and migrants ($\overline{W}_M$) are all at time t (the subscript t is dropped from all equations).

Adaptation is controlled by a single locus with alternative alleles A and a, which are found at frequencies p and $1 - p$ on the island and P and $1 - P$ on the continent. The frequency of A on the island will change from one generation to the next according to

$$p_{t+1} = \frac{[p^2 W_{AA} + p(1-p)W_{Aa}]n + [P^2 W_{AA} + P(1-P)W_{Aa}]MN}{\overline{W}_R n + \overline{W}_M MN} \tag{A2}$$

where W_{AA} and W_{Aa} are the fitness of AA homozygotes and Aa heterozygotes (generally, W_{ij}). To maintain additivity, W_{Aa} was always the average of W_{AA} and W_{aa}. The mean fitness of residents and migrants is given by

$$\overline{W}_R = p^2 W_{AA} + 2p(1-p) W_{Aa} + (1-p)^2 W_{aa} \tag{A3a}$$

and

$$\overline{W}_M = P^2 W_{AA} + 2P(1-P) W_{Aa} + (1-P)^2 W_{aa} \tag{A3b}$$

The above equations show how population size and allele frequency change on the island as a function of the fitness of different genotypes. The final step is to generate realistic population growth by making the fitness of each genotype a function of the number of individuals on the island. That is, the absolute fitness of a given genotype should decrease as the population size increases (i.e. density dependence). One way to incorporate this effect is to express the fitness of each genotype ij (W_{ij}) in equations (A2), (A3a) and (A3b) as

$$W_{ij} = x_{ij} \exp[- y_{ij} n] \tag{A4}$$

where x_{ij} gives the fitness of genotype ij when the population size is far below the carrying capacity (i.e. when n is very small, $W_{ij} = x_{ij}$) and y_{ij} gives the strength of density dependence acting on genotype ij. When $y_{ij} = 0$, density dependence is absent ($W_{ij} = x_{ij}$). As y_{ij} increases, density dependence becomes stronger.

Quantitative-genetic model

As in the single-locus model, population size on the island (n) changes according to equation (A1), but now the fitnesses of residents and migrants are a function of their phenotypes, rather than their allele frequencies. The fitness of an individual with phenotype z, when the population size on the island is n, is given by $W(z,n)$. As in other quantitative-genetic models, I assume that z is normally distributed with a mean of $\bar{z}$ and a variance of V on the island ($Norm(z; \bar{z}, V)$) and a mean of $\overline{Z}$ and a variance of V on the continent ($Norm(z; \overline{Z}, V)$). The mean fitness of residents is then

$$\int_{-\infty}^{+\infty} Norm(z; \bar{z}, V) W(z, n) dz \tag{A5a}$$

and the mean fitness of migrants is

$$\int_{-\infty}^{+\infty} Norm(z; \overline{Z}, V) W(z, n) dZ \tag{A5b}$$

To calculate the mean fitness of residents, I specify $W(z,n)$ as

$$W(z, n) = \alpha \exp\left[-\frac{(z-\theta)^2}{2\omega^2}\right] \exp\left[-\frac{n}{c}\right] \exp\left[-\frac{(z-\theta)^2}{2k^2} n\right] \tag{A6}$$

In this equation, the first term (α) is a constant that represents the fitness of a perfectly adapted individual in the absence of density dependence (i.e. when population size is very small). The second term represents how fitness is influenced by a given deviation of an individual's phenotype (z) from the optimal phenotype (θ) under a given strength of stabilizing selection around the optimum (width of the fitness function: smaller ω reflects stronger stabilizing selection). This term reflects the effect of adaptation in the absence of density dependence. The third term represents the effect of density dependence on fitness in the absence of an interaction with adaptation. Here, c is a

constant that dictates the strength of density dependence: as c decreases, a given population size has a stronger effect on reducing fitness. The fourth term represents the interaction between adaptation and density dependence, where k is a constant that determines how much a given phenotypic deviation from the optimum is negatively impacted by population density (smaller k reflects a stronger impact). This interaction term is important because it allows different phenotypes to be differentially influenced by population density. Through this term, a given increase in population density can have different effects on migrants and residents – because they differ in mean phenotype.

Substituting equation (A6) into equation (A5a), and integrating, yields the mean fitness of residents:

$$\overline{W}_R = \alpha \frac{\exp\left[\frac{-(\bar{z}-\theta)^2}{2(V+A\omega^2)}\right]\exp\left[-\frac{n}{c}\right]}{\sqrt{1+V\left[\frac{n}{k^2}+\frac{1}{\omega^2}\right]}} \tag{A7}$$

where $A = k^2/[k^2 + n\omega^2]$. The mean fitness of migrants is then obtained by substituting the equivalent version of equation (A6) into equation (A5b), again yielding equation (A7), except that $\bar{z}$ is replaced with $\bar{Z}$, which remains constant.

The mean phenotype on the island will change from one generation to the next according to

$$\Delta\bar{z} = m\bar{Z} + (1-m)\bar{z} + G\beta \tag{A8}$$

where m is the proportion of individuals on the island that are migrants from the continent, G is the additive genetic variance for the trait, and β is the selection gradient acting on the trait (for the derivation, see Hendry *et al.*, 2001). As in the single-locus model, m can be expressed relative to population sizes and the probability that a continent individual migrates to the island – that is, $m = (MN)/(MN + n)$.

As the trait evolves towards its optimum on the island, the selection gradient changes. Under such conditions, the standard approach is to express selection gradients as a function of (1) the deviation of the mean phenotype ($\bar{z}$) from the optimal phenotype (θ), (2) the phenotypic variance (P), and (3) the strength of density-independent stabilizing selection around the optimum (ω). That is, $\beta = -(\bar{z} - \theta)/(\omega^2 + P)$ (e.g. Via and Lande, 1985; Hendry *et al.*, 2001). I here adopt the same approach but incorporate the following modifications. First, selection in the present model acts on the mean phenotype *after* migration; hence $\bar{z}$ is replaced with $m\bar{Z} + [1-m]\bar{z}$ (Hendry *et al.*, 2001). Second, stabilizing selection depends not only on density-independent selection (ω) but also on density-dependent selection (see above), which is a function of density (n) and the effect of density on a given phenotypic deviation from the optimum (k). After taking these effects into account, the selection gradient as obtained in the standard fashion is:

$$\beta = \frac{-[(m\bar{Z} + [1-m]\,\bar{z}) - \theta]}{A\omega^2 + P} \tag{A9}$$

where A is defined as above.